A Complication of Pyloromyotomy

Recovery After Perforation of Duodenum

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ACCIDENTAL PERFORATION of the duodenal mucosa during Fredet-Ramstedt pyloromyotomy for hypertrophic pyloric stenosis is the most common and most feared complication of this operation. The reported incidence ranges from as low as one in 385 operations[1] to as high as 28 in 110 cases.[3] It is not a serious accident if the opening is seen when it is made and is sutured.[2] However, if not discovered, the continuing escape of duodenal contents will almost invariably result in fatal peritonitis. In a review of the literature no report was found of a proved case in which a patient survived unrecognized perforation. The purpose of this report is to present the case of an infant with unrecognized perforation who survived.

REPORT OF A CASE

The patient, a male infant, was operated upon elsewhere for hypertrophic pyloric stenosis at two months of age. The usual Fredet-Ramstedt procedure was done, permitting the mucosa to bulge into the wound. A small venous bleeding point at the duodenal end of the incision was noted but was not ligated. The highest temperature was 102° F. on the first postoperative day. The patient was discharged on the fifth day with a normal temperature and retaining feedings. No antibiotics were administered during the hospital course.

The infant was admitted to the Los Angeles County Harbor General Hospital 11 days after the operation. The mother stated that four days previously (two days after discharge from a private hospital following the above described operation) the patient began to vomit infrequently and in small amounts. The abdomen became increasingly distended and no bowel movements were noted. Vomiting increased in frequency and the vomitus, at first bile-stained, later became thick, greenish-yellow and had a "bitter odor."

The patient was extremely emaciated and dehydrated, and the pattern of hugely distended loops of bowel was visible through the anterior abdominal wall. No masses, rigidity or tenderness were noted. Bowel sounds were infrequent and high-pitched. No abnormality was noted in rectal examination. Results of laboratory examinations of the blood and urine were within normal limits. A roentgenogram of the abdomen showed several parallel loops of greatly distended bowel with an appearance compatible with mechanical obstruction of the small bowel.

At operation, with the patient under local anesthesia, greatly dilated loops of small bowel were observed. In the right subhepatic space there was an abscess, containing approximately 4 cc. of bile-stained pus, that was connected with a 2 mm. opening in the duodenum at the distal end of the partially healed pyloromyotomy wound. Bile-stained fluid was easily expressed from the perforation, which was closed with two through-and-through sutures of No. 00000 gastrointestinal chromic catgut. Fibrinous peritonitis involved all the right peritoneal gutter and many loops of adjacent small bowel. The point of obstruction was about 6 inches proximal to the ileocecal valve. The obstruction was relieved by sharp and blunt dissection and the dilated bowel was decompressed by suction enterotomy. It then became evident that a segment of ileum was not viable. Eighteen inches of nonviable and denuded ileum were excised. An open end-to-end anastomosis was done, using an inner row of No. 00000 chromic catgut and an outer layer of interrupted Lembert sutures of No. 00000 silk. The bowel was returned to the peritoneal cavity and, in the belief the patient was near death, the abdomen was hurriedly closed with through-and-through retention sutures of No. 30 steel wire.

That the patient lived was attributable largely to excellent postoperative care by the pediatric staff. Convalescence was retarded because of healing of the skin incision by second intention. By the twenty-second postoperative day the patient had gained 2 pounds in weight and was getting along satisfactorily. On the forty-third postoperative day he was discharged. The body weight then was 10 pounds. At the age of seven and a half months it was 18 pounds and the patient was apparently eating and developing as any normal child.

COMMENT

Perforation in this case was discovered only because intestinal obstruction developed. In this instance it may be significant that there was bleeding at the distal end of the Fredet-Ramstedt pyloromyotomy site during the original operation. Szilagyi and McGraw[4] pointed out that there is a deep artery and vein running across the duodenal end of the incision at the fornix of the duodenal mucosa, thus indicating the danger area. The hypertrophied pyloric musculature extending into the duodenal lumen can be likened to the uterine cervix extending into the vaginal canal. Consequently, the incision into the hypertrophied pylorus can easily nick the duodenal mucosa if carried too far distally or too deeply. If there is any suspicion of mucosal injury, attempts should always be made to discover them at the time of operation. There are three methods of demonstrating such perforations: (1) simple observation of the hole in the mucosa emitting bubbles of bile-stained fluids; (2) compression of the stomach and duodenum in an effort to force gas or fluid through a minute perforation; (3) with the duodenum compressed injection of air or methylene blue through a catheter in the stomach in an effort to force fluid or gas through the minute perforation. The use of a binocular loupe may aid vision in this area. Once the perforation is found, simple closure with fine catgut or silk sutures on an atraumatic needle solves the problem. Other methods have been suggested but are probably unnecessary.

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The infant in the present case survived three highly lethal mechanisms — duodenal perforation, intestinal obstruction and small bowel resection. The present-day medical armamentarium of scientific management of fluid and electrolyte balance, antibiotics, blood transfusions, trained anesthetists, and pre- and postoperative care by pediatricians encourages surgeons to intervene in seemingly hopeless problems with some expectation of success.

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REFERENCES

1. Donovan, E. J.: Congenital hypertrophic pyloric stenosis, Lewis' Practice of Surgery, Vol. VI, Chap. 7, p. 12.

2. Norris, W. J.: Congenital hypertrophic pyloric stenosis, West. J. Surg., Gyn. & Obst., 41:377, July 1933.

3. Person, E. C.: Congenital hypertrophic pyloric stenosis, Surg. Clin. N. Am., 30:529, April 1950.

4. Szilagyi, D. E., and McGraw, A. B.: The problems of infantile pyloric stenosis with particular reference to surgical treatment, Surgery, 13:764, May 1943.

Sarcoidosis

A Diagnostic Problem

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BOECK'S SARCOID is usually considered a benign, self-limited disease that in most cases leaves little if any residual damage. Often the disease is entirely asymptomatic and is detected fortuitously on routine x-ray examination of the chest. In the present case, however, sarcoidosis presented a diagnostic problem in an acutely ill patient.

During an acute febrile illness with definite or indefinite evidence of abnormality in the chest on x-ray examination, it is important to consider the following in differential diagnosis: (1) virus pneumonitis; (2) bacterial pneumonitis; (3) Hodgkin's disease; (4) bronchiogenic carcinoma; (5) collagen disease; (6) tuberculosis; (7) granuloma (Boeck's or Wegener's). Although it is well known that usually in sarcoid disease there is involvement of the hilar nodes and lungs, the diagnosis cannot be made from roentgenologic observations alone, for Boeck's sarcoid can simulate pulmonary disease of almost any kind. Laboratory findings that aid in making the diagnosis are: (1) elevation of serum protein and globulin values; (2) accelerated sedimentation rate; and (3) a negative reaction to tuberculin. Elevation of the blood calcium level and increased alkaline phosphatase activity have been reported in a few cases but are not constant findings. Of course, if there are skin lesions or peripheral node enlargement, biopsy of material from these lesions is the most accurate method of diagnosis. The Kveim antigen injected intracutaneously is highly specific for sarcoidosis, its only disadvantage being the length of time required for a nodule to form and then for biopsy. When the diagnosis is uncertain and no peripheral nodes are present, needle biopsy of the liver may give positive evidence of sarcoidosis.

REPORT OF A CASE

A white man 58 years of age was first observed February 10, 1953, with complaints of morning and afternoon fever (101° F.), considerable nausea, lack of appetite and "cigarette" cough—all of about three weeks' duration. (He stopped smoking soon afterward and the cough disappeared.)

Upon examination, tachycardia was noted and the temperature was 100.6° F. Fluoroscopic examination of the chest was carried out and a fairly well defined strand of density extending from the lower hilar area to the periphery of the right lower lobe was observed. The blood sedimentation rate (Linzenmeier) was 18 mm. in 30 minutes. Penicillin was given, 600,000 units a day for a period of one week, and during that time the temperature reached 104° F. on several days, associated with chills. As the patient refused to enter the hospital for further study, penicillin was discontinued and aureomycin, 1 gm. per day, was administered. The patient felt worse and had considerable nausea. In the afternoons and evenings the body temperature rose to 102° F. to 103° F. During a two-week period the abnormalities noted were a few dry rales at the right lung base posteriorally, persistent tachycardia, 4 plus albuminuria with granular casts and accelerated blood sedimentation rate. An additional patch of density originating near the left hilum and extending toward the left base was observed fluoroscopically. At no time was any degree of dyspnea noted, although on several occasions the lips were cyanotic.

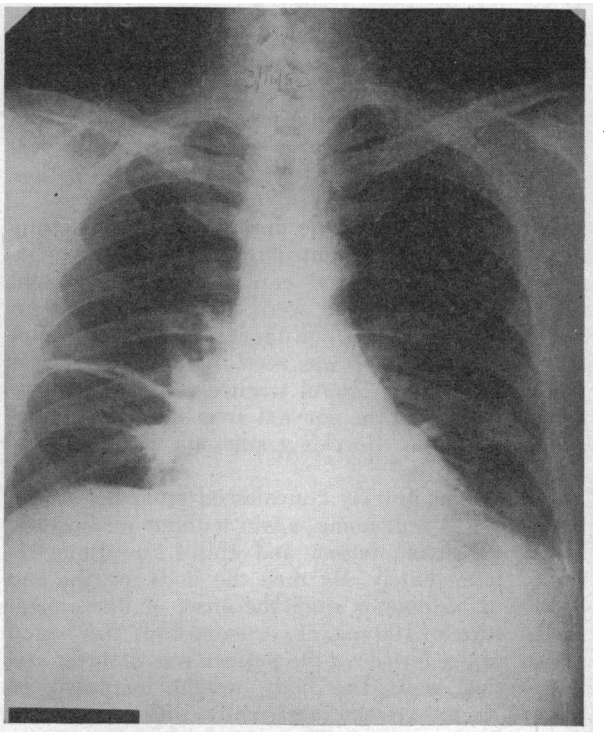

Figure 1.—Linear strand of density extending from right hilum to periphery, with beginning involvement of left hilar area.